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ABSTRACT

The use of a traditional syndrome-classification system for aphasics is examined critically from the different perspectives of medicine and psychometrics. Medicine views syndromes as dichotomous (present or not present) and necessarily indicative of an underlying pathognomic state or process, and psychometrics sees performances as varying along a continuum. The issues of the pragmatic value of syndromes, or their correctness versus their fruitfulness, and the procedures for identifying syndromes are discussed in the context of recent research. The use of distinctive recovery patterns as a means of classifying aphasics and determining the appropriate therapeutic approach for each is proposed and illustrated with a model. (MSE)

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APHASIA SYNDROMES AND THEIR RECOVERY

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1. INTRODUCTION

Throughout the history of the study of aphasia, some sort of system for classifying patients has been used. It is useful initially to characterize different approaches and ask whether or not a syndrome-classification system is still useful in research on aphasia.

An important dichotomy in thinking about classification can be characterized by the terms medical vs. psychometric. The medical view of syndromes is that they are characterized by combinations of symptoms (signs) that are either present or not present. The patient is either anomic or not anomic, apraxic or not apraxic etc.

The psychometric view sees performances as varying along a continuum. Combinations of performance measures gives a performance profile. Arbitrary cut-off points may be used to define syndromes, but statistical techniques for detecting groupings of scores are needed to justify a classification system. The dimensions used for classification are more basic that the syndromes themselves.

The medical and psychometric views also entail a difference in whether syndromes are seen as "real" entities or as convenient fictions. In the medical view a syndrome should be indicative of an underlying pathognomic state or process.

A viable research strategy in aphasiology until recently has been to ask what linguistic deficit is shared by all patients with a clinically defined type of aphasia. There is a recent debate pointing out that all the defining features of a syndrome are rarely present. There is thus only a "family resemblance" between different members of a syndrome. Asking the question "what is the mechanism producing the deficit in Broca aphasia, is it prosodic or syntactic" may thus be meaningless since it presupposes a real core deficit in Broca aphasia which does not in fact exist. Some neurolinguists (Schwartz 1984) have drawn the conclusion that syndromes are therefore uninteresting objects for research.

Poeck (1983) has argued that although syndromes are fictions they are useful fictions, also for neurolinguistic research. Other neurologists, like Kertesz (1979) seem to agree that so long as a classification system allows you to make predictions

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about lesion location or prognosis, it is useful in itself. These views reflect a psychometric view of syndromes.

The procedure for identifying syndromes also reflects the conflicting views. Some authors take for granted that there are "real" cases of Broca aphasia, Wernicke aphasia etc., and the trouble is to place a given patient on the map. This may be done intuitively or by a statistical procedure. On the other hand the approach of Kertesz (1979) is taxonomic, which means that he is willing to define a system with sharp boundaries and specific classification rules. The problem "I think this patient is a Broca aphasic, but is he really?" has meaning in the first context, but not in the second because Broca aphasia is fully defined by the classification rules. In the second case it is not the correctness, but the fruitfulness of the classification which is at issue.

In the current literature there thus seems to be a division between neurolinguistically oriented researchers who now want to abandon the classical aphasia syndromes as theoretically uninteresting and clinically oriented researchers, who point out their pragmatic value. The disagreements on classification reflect in my view to a large extent a contrast between a medical and a psychometric view of syndromes. My own view is that syndromes are hypothetical entities which must be defined psychometrically. The fruitfulness of a proposed set of entities and classification rules for research and clinical work must be validated against specified alternatives on a competitive basis. In the further discussion I address two more general questions:

1. Which syndromes should be regarded as valid and how many are they?
2. Assuming that syndromes are clinically useful fictions, are they also theoretically interesting?

2. BACKGROUND

The Institute for Aphasia and Stroke was created by the National Association for Public Health (a private fund) in 1973. The institute was taken over by Sunnaas Rehabilitation Hospital in 1977, but with continued support from the National Association. A program for testing patients was developed and material has been continually registered over a period of about 7 years.

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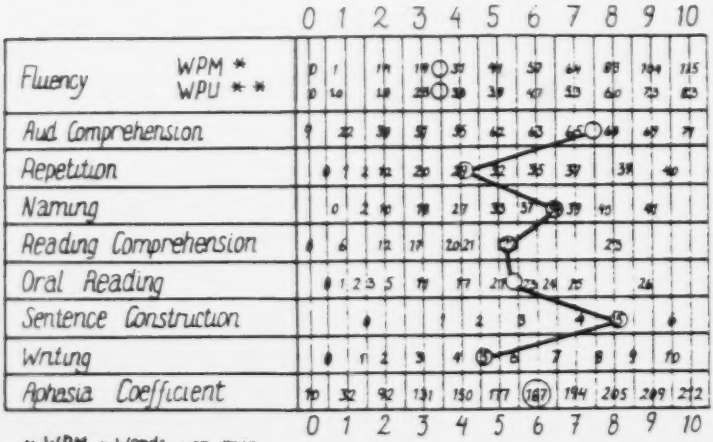
Subjects: 249 aphasic patients have been tested over a period of time between 1977 and 1982. About 130 patients have been followed with repeated testing. The patients are predominantly vascular cases. As far as possible all cases admitted to the hospital with aphasia were tested.

3. CLASSIFICATION

Aphasics are tested with Norsk Grunntest for Afasi developed and standardized in our clinic (Reinvang and Engvik, 1980; Reinvang, 1985). The test includes rating of spontaneous speech, tests of auditory comprehension, repetition, naming, reading comprehension, reading aloud, sentence construction, and writing. The results are transferred to a percentile scale, based on the distribution of scores in a normative group of 161 patients.

Based on statements in the literature and criteria suggested in other tests, definitions with exact quantitative criteria are given for all the classical syndromes in the Wernicke-Lichtheim model. The Wernicke-Lichtheim system of classification has 8 major syndromes and several rare ones. It has been adopted by most research



* WPM = Words per min.
** WPU = Words per utterance

Figure 1. Aphasia test profile in a Broca aphasic.

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centers on aphasia (Kertesz, 1979; Goodglass and Kaplan, 1972). In the present adaptation of the system Broca aphasia, for example, is defined as non-fluent speech with auditory comprehension and naming more than 20 percentile points higher than fluency. The following test profile satisfies the criteria for a case of Broca aphasia.

The system is not exhaustive, and unclassifiable cases occur. The view implicit in this procedure is that aphasia types are hypothetical constructs, and that any proposed classification must be evaluated. An attempt was made by my colleagues Sundet and Engvik (1985) to use a statistical procedure (clustering analysis) to evaluate the proposed classification system.

Included in the study were 193 aphasic stroke patients seen at median time since onset of 216 days (range 40 days-7,5 years). The sex ratio was 122 male to 71 female and mean age was 56.5 years (range 22-80 years). Input variables selected for cluster analysis were Fluency, Comprehension, Repetition, Naming and Communication Capacity. Program 2M in BMDP was used with the Euclidian distance measure and the centroid linkage algorithm for joining clusters as options. A discriminant analysis with the same input variables was used to check the validity of the clusters.

The procedure yields nine distinct clusters and the discriminant analysis shows that two variables can be used to predict the cluster classification with a success rate of 85 %. The first variable combines the measures of Communication Capacity, Comprehension, Repetition, and Naming, whereas Fluency alone formed the second variable.

This analysis suggested to Reinvang (1985) that aphasia types can be reduced to four major categories with a range of severity within each category. This view is in accordance with that of the Aachen group who conceive of four major aphasic syndromes classifying about 80 % of aphasic stroke patients (Poeck, 1983).

For further analysis Reinvang (1985) used the following system (Table 1). The entries in each cell can be conceived as types of aphasia, whereas the marginal terms are the dimensions defining the types.

A traditional argument for using the more detailed Wernicke-Lichtheim system is its alledged ability to predict lesion localization with significant success (Kertesz, Harlock and Coates, 1979). Comparing it to the less detailed classification system just described, I have tested the ability of the two systems to predict lesion localization, and found them to be quite similar. In both cases the relation of function to localization is a probabilistic one. The clinico-pathological correlations give no strong indications for accepting the more differentiated classification system. The analyses are reported in Reinvang (1985, chapter 7).

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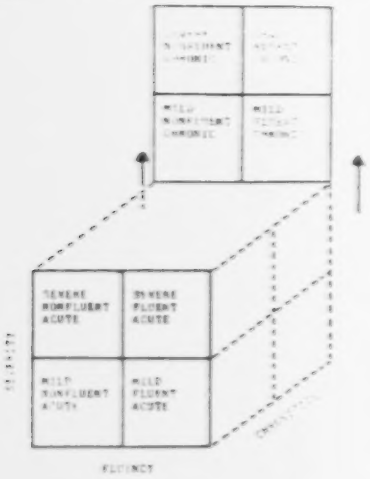
Table 1. System for grouping of aphasics.



4. RECOVERY

Besides the classically accepted statement that aphasia type predicts lesion localization, what else are syndromes useful for?

Statements about prognosis in aphasia are notoriously difficult to evaluate. If you mean the prognosis of full recovery, then it is surely poor (Reinvang, 1984). If you mean predicting the end state, then surely the most significant factor is the severity of aphasia initially. Severe aphasics are likely to remain more severe than mild aphasics. The statement that prognosis is better in Broca aphasia than in global aphasia is relatively trivial if it means only that relative rank order of severity is usually preserved in recovery.

Is anything known about the magnitude of the recovery, i.e. the difference between the initial and final state? This question raises problems of methodology because difference scores are notoriously unreliable. I note, however, that aphasia test scores show very high coefficients of reliability as measured with coefficients of internal consistency, so that I do not think the results reported here can be dismissed on purely statistical grounds (see also Willmes, 1985).

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Using the cube model of Table 1, I have asked which dimensions of the classification predict magnitude of improvement (test-retest difference in the measure of degree of aphasia, the AC). Two time periods are considered, the acute (< 6 months) and the chronic (> 6 months). Analysis of variance is a statistical method that can be used to evaluate this question and it yields the following results in 134 patients (Table 2).

Table 2. Recovery as a function of subgroup.

Group variable	F	p
Type (nonfluent vs. fluent)	1.43	n.s.
Severity (low performance vs. high)	14.27	.001
Chronicity (acute vs. chronic)	5.71	.02

Adapted from Reinvang (1985), p. 141.

All the dimensions of the classification except fluency are the source of significant main effects. Acute patients improve more than chronic and severe patients improve more than mild, when unadjusted difference scores are analyzed. The fact that Kertesz (1979) comes to different conclusions can be explained by his use of recovery scores expressing a ratio of absolute change to maximum possible change.

Analysis of variance has the possibility of analysing for interaction effects. This is the interesting point in regard to syndromes, because a syndrome (like mild, fluent and chronic) in this context is an interaction, or a special combination of values, of the basic dimensions.

Four two way interactions were found, indicating that distinctive subgroups behaved differently compared with the general recovery trend. Some interesting patterns can be pointed out.

The fluents make a much better recovery when the first test is in the acute than in the chronic stage, whereas for the non-fluents this is only a slight tendency. Recovery is thus less dependent on time after injury in non-fluents, as has also been found by Sarno and Levita (1979).

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The severely aphasic patients make a high recovery score when the first test is in the acute stage and a poor score in the chronic stage. The mild aphasics make about the same recovery regardless of time after lesion. Recovery is thus less dependent on time after injury in mildly aphasic patients.

5. PROGNOSTIC SIGNIFICANCE OF NEUROPSYCHOLOGICAL FINDINGS

To further evaluate the hypothesis of distinctive recovery patterns, information about results on other neuropsychological tests is introduced.

All aphasics are tested with a standard neuropsychological battery. It is a mixture of standard psychometric tasks, tests from well known neuropsychological batteries and tests from the experimental neuropsyhological literature (Table 3.)

Table 3. Overview of neuropsychological tests.

Function	Problem area	Test
	Verbal immediate memory	Digit span Pointing span
LANGUAGE	Verbal learning	Supraspan learning
	Verbal conceptual function	WAIS-Verbal IQ Paired associate learning
	Ideational apraxia	Object use
MOTOR FUNCTION (APRAXIA)	Ideomotor apraxia	Movement imitation
	Motor coordination	Grooved pegboard Finger tapping
	Visual immediate memory	Corsi blocks
VISUAL ABILITIES	Visual route learning	Supraspan Corsi blocks
	Problem solving	Raven CPM WAIS-Performance IQ
	Construction	Copying

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On the basis of factor analytic studies (Reinvang & Sundet, 1985) my present view is that aphasia is a multi-dimensional neuropsychological deficit in which language, motor performance and visual abilities are identifiable dimensions. Are the non-verbal dimensions of aphasia prognostically significant with respect to recovery of language? Since recovery varies in subgroups, the analysis follows the classification system already proposed.

A summary of the results of these analyses is given (Table 4). The fluent versus nonfluent distinction is omitted because this variable showed no main effect on recovery. The groups defined by the significant interaction terms were not analyzed because the number of subjects in each group was small.

Table 4. Prognostic neuropsychological signs.

Subgroup	Predictive function or problem area
SEVERE	Motor function (ideational apraxia, ideomotor apraxia, and coordination))
	Verbal immediate memory
	Visual immediate memory
MILD	Construction
	Ideational apraxia Ideomotor apraxia
	Visual route learning
ACUTE	Verbal immediate memory
	Ideomotor apraxia
CHRONIC	Ideomotor apraxia Coordination

Adapted from Reinvang (1985), p. 143.

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Correlations derived by such an explorative procedure should be interpreted with reserve. If we bear in mind the factor analyses of neuropsychological tests, it is reasonable to require that several tests representative of a factor show significant correlations before this factor is taken to have a significant prognostic value.

Using the factor analyses as guidelines for interpretation, one may conclude that apraxia was a prognostic sign in both severe and mild aphasics. The prognostic relation had the opposite sign in the 2 groups, and therefore the effects cancelled each other out in an overall analysis.

Verbal immediate memory (represented by two tests) was also a prognostic sign in severe aphasia but not in mild aphasia.

The visual non-verbal tests do not appear to be strongly correlated with prognosis although scattered significant correlations are found.

The clearest prognostic implications of neuropsychological functions appeared in relation to the severe vs. mild distinction. The findings are not so readily interpretable with respect to the acute versus the chronic distinction. Low but significant correlations on a scattered selection of tests are difficult to assess, but the general point of subgroups is supported.

6. RELATIONS BETWEEN FUNCTIONS AT DIFFERENT STAGES OF RECOVERY

Is the functional domain measured by the different tests differently organized in acute and chronic patients? The question is not about the content, but about the structure of the preserved functions. To answer it a factor analysis of the aphasia test results and the neuropsychological tests is performed. Consider a hypothetical contrast between two possible modes of functional organization.

Case 1: The brain is a single channel of processing, and all tasks are performed by the same neurological and functional devices.

Case 2: The brain has a number of well differentiated processing mechanisms that can perform different functions.

If studied with factor analysis case 1 should be functionally characterized by a single factor accounting for all the variance in performance. Case 2 should show several functional factors and absence of a prominent G (or general) factor.

When applied to the severe or mild, and chronic or acute patients, the pattern observed is that severe and acute patients show a smaller number of different factors and a higher G-factor, whereas the mild and chronic show a higher number

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of different factors and a smaller G-factor (see Reinvang 1985, chapter 8). It was thus the severely aphasic group that showed the clearest trend in the direction of greater differentiation of function with time.

Summarizing the results on recovery I found that the severe aphasics made a greater-than-average recovery, especially if, in addition to being severe, they were in the acute stage and young. Several neuropsychological variables representing factors of apraxia, verbal memory, and nonverbal memory were predictive of recovery. Finally, the severe aphasics showed signs that a process of functional differentiation was taking place between the acute and the chronic stages of the illness.

The finding of distinctive patterns over time and across functions may indicate different recovery mechanisms in different subgroups. Because the differences between groups were not dramatic, it is more reasonable to conclude that several recovery mechanisms may have operated simultaneously, but with different weigths in different groups.

7. DISCUSSION

What theoretical conclusions (or speculations) can be advanced? In his influential review and critique of current classification approaches, Caramazza (1984) considers problems for the view which he himself advocates, namely that well motivated theories of normal performance should be the basis for analyzing pathological phenomena. The **fractionation** assumption is "the belief that brain damage can result in the selective impairment of compontents of cognitive processing" (p. 10). He says that "a less than optimistic view of the interpretation of pathological performance is that even if the fractionation assumption were to be true, the remaining, unimpaired processes will work differently when one component is not functioning normally. In this case the pathological performance would not have a transparent relation to the working of the normal system, and would make the analysis of pathological cases irrelevant for the understanding of normal cognition" (p. 11). He then goes on to dismiss this possibility by an act of faith, since it is threatening to his whole program.

I think the cognitive-linguistic approach to neuropsychology is in general fruithful, but not sufficient as a theoretical model. Let us ask if Caramazza's problem does not admit of a solution which does not invalidate his approach.

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Some now almost forgotten classical papers on aphasia focus on the qualitative changes in cerebral function taking place after injury. I have in mind the work of Goldstein and his concept of loss of differentiation (dedifferentiation) after injury (Goldstein, 1948). The resulting effect on behavior is concreteness, rigidity, and stimulus bound responding. Although he goes too far in generalizing this principle, I think it should be brought back into our theories of aphasia. In a large cerebral lesion producing a severe degree of aphasia, my results can be taken to show that a closer linkage (loss of differentiation) exists between language and non-verbal neuropsychological functions. Other clinical signs of this loss of differentiation are the context-dependency of the patient's responses and his inability to process two things at the same time.

Some functional interpretations may be suggested for the three dimensions I have used to define the aphasic condition.

The fluency dimension is related to a specific kind of information processing mechanism. There seems to exist a localized and functionally distinct mechanism for maintaining normal fluency of speech. Injury to this mechanism produces nonfluency. Except for fluency I don't think we have any strong indication that other aphasic phenomena reflect processing mechanism in any transparent way. I specifically do not think that auditory comprehension, as measured in tests of aphasia, reflects any well defined linguistic processing mechanism with a neurological correlate.

The severity dimension must of course reflect failure of specific processing mechanisms, but also the loss-of-differentiation aspect in which the brain becomes more like a single channel device. The multi-dimensional nature of severe aphasia partly reflects the undifferentiated response of preserved brain areas to varying functional demands.

Chronicity is linked with the kind of recovery mechanism which is operating. One class of such mechanisms is the relearning or reorganization processes. These are similar in that both presuppose a stable underlying processing system, but one focuses on strategies whereas the other does not try to analyze the system. This class of mechanisms is operative in chronic aphasia.

The other class of processes, which are more active in acute aphasia, contains relocalization and what I will call re-differentiation of function. This supposes that some degree of readjustment of the function-to-structure relationship takes place. The idea that one area of the brain takes over the function of another is, I think, biologically unmotivated. My final exercise will be to show an example of how re-

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differentiation can operate to establish a partly new pattern of cerebral organization without involving ideas of arbitrary shuffling around of functions.

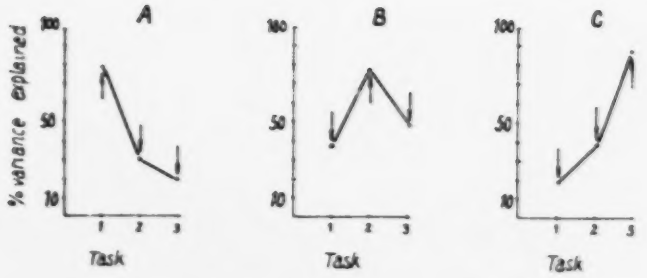

Figure 2. Differentiation of function in three areas (for explanation, see text).

Figure 2 represents a hypothetical system consisting of three devices with different, but overlapping functional profiles. The devices can be seen as schematic representations of brain areas. The three devices communicate with each other, and as a result of this interaction the functional profiles are sharpened in the direction that functions which one device performs better than any of the others are boosted, and less efficient functional capacities are supressed. With an injury to one of the devices in such a system the basic functional profile of the uninjured devices would remain unchanged. It is easy to imagine, however, that part of the functional profile of a remaining device might now be boosted rather than suppressed because it is the most efficient device with respect to that function in the residual system. This argument is pursued in greater detail in Reinvang (1985, chapter 9).

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These then are the kinds of recovery processes at work in chronic and acute aphasics. If we knew more about how to stimulate them, we might come closer to a rational therapeutic approach. This is especially needed in relation to the severe aphasics, a group which in my clinical judgement does not profit well from therapy based exclusively on an information theoretical componential analysis of their deficit.

I do not share the fear of Caramazza that accepting the sort of model I have suggested will invalidate the componential approach to the study of information processing. We study phenomena along a continuum, and there is no sharp breaking point at which we must throw up our hands in dispair. If we can study cognition in non-human species then it surely cannot be a precondition for the study of aphasics that they can be modeled as normals-minus-one. The analogy intended is to music-minus-one, which is a recording of a concerto without the solo instrument. Without carrying the analogy too far I suggest that in applying it to loss of language after brain injury the resulting concerto becomes rather a different piece with the other instruments having to adjust their functions without loosing the organizing perspective of producing music.

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